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Friedlander Bacillus Meningitis

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FRIEDLANDER bacillus meningitis is rare. Thirty-three cases have been reported in the literature.[4, 6, 8, 9] In a recent report, Pain and his co-workers[7] note a case of Friedlander bacillus, type A, meningitis, with the organism secured from cerebrospinal fluid, blood and aural discharge. The patient received sulfadiazine, penicillin, and streptomycin, but died 33 hours after the first dose of streptomycin. In almost all cases there has been coincidental septicemia, and almost all the patients have died. The disease was found to originate in the lung in five of 29 cases reviewed by Ransmeier.[8] In 1943, 51 cases of primary Friedlander bacillus pneumonia were reported,[5] and among them were two cases of Friedlander bacillus meningitis secondary to Friedlander pneumonia.

CASE REPORTS

CASE 1. The patient (Case 9 of the Friedlander pneumonia series)[5] was a 52 year old white male. He entered the hospital in a preterminal comatose state, and died in seven hours. Friedlander bacilli, type A, were cultured from the sputum, blood, and cerebrospinal fluid. The patient had pneumonia involving the right upper lobe and the left lower lobe, and also meningitis. He died without receiving any specific therapy. (Pre-chemotherapy era.) At autopsy, pneumonia and purulent meningitis were the chief findings. Postmortem cultures of the lung, meninges, and spleen were positive for Friedlander bacilli, type A.

CASE 2. The patient (Case 50 of the Friedlander pneumonia series)[5] was a 37 year old white male. He entered the hospital acutely ill, with cough and thick, tenacious, mucopurulent, blood streaked sputum. Examination and a chest roentgenogram revealed pneumonia of the entire left lung and, in addition, signs of meningitis were present. Friedlander bacilli were cultured from the sputum, blood and cerebrospinal fluid. No typing of the organism was done. The patient died on the fourth hospital day after an acute toxic course. No chemotherapeutic or anti-biotic agents were available (1935). Postmortem examination revealed a Friedlander pneumonia and purulent meningo-encephalitis. Postmortem cultures of the heart, lung, blood, spleen and meninges were positive for Friedlander bacilli.

As in most of the reported cases of infection with Friedlander bacilli, neither of the patients received specific therapy. Two patients given sulfonamides are reported to have recovered.[8, 9] One patient treated with streptomycin died, but "the absence of gross and the nearly complete absence of microscopical evidence of meningitis at autopsy are stressed."[9] Experimental evidence indicates that both sulfadiazine[1] (Table 1) and streptomycin[2, 3] are effective in vitro against certain strains of Friedlander bacilli, although not against all.

TABLE 1.—*Mice Survival and Chemotherapy*

(Adapted from Feinstone, Williams, Wolff, Huntington, and Crossley[1])

	No. of Mice	Mice Surviving	Per Cent Survival
Sulfanilamide	100	2	2.0
Sulfapyridine	99	6	6.0
Sulfathiazole	100	2	2.0
Sulfadiazine	86	63	73.6
Controls	78	0	0.0

Heilman[2] studied nine strains. Four of these, type A, had not been recently isolated. Five strains had been recently isolated, but were not type A or B. All nine strains were sensitive to streptomycin in vitro, the recently isolated strains being more sensitive. Using a single strain of Friedlander bacilli sensitive to streptomycin, Heilman found that in mice inoculated intra-abdominally with 10,000 times the lethal dose of bacilli, streptomycin given three hours after the inoculation and then for a total of three days, would give complete protection. The control mice, not receiving streptomycin, were all dead within one day. Similar experiments in which mice were inoculated intra-abdominally with 1,000 times the lethal dose of a type A strain of Friedlander bacilli, and then were treated with streptomycin suspended in oil and beeswax, produced essentially similar results. When the mice were infected by intranasal doses of Friedlander bacilli type A, the protection given by streptomycin was less, although here too the drug was of definite value. The mortality rate for streptomycin-treated intranasally infected mice (seven days of therapy) was 13 per cent compared with 100 per cent for the control group. It is important to stress that streptomycin was given very soon (three hours) after the introduction of the Friedlander bacilli (Table 2). It is clear that adequate therapy had best include both sulfadiazine and streptomycin, to be given promptly and in adequate doses, and to be pursued vigorously. These drugs can be of value only before suppuration, or at least extensive suppuration, has begun. Their greatest value is, of course, at the onset of the disease. The importance of an adequate and intensive early search for bacterial invaders (smears and cultures of sputum and cerebrospinal fluid and blood cultures) is clear.

SUMMARY AND CONCLUSIONS

1. Two cases of Friedlander bacillus meningitis are reported. Both patients died, one in seven hours and one on the fourth hospital day. Sputum, blood, and cerebrospinal fluid cultures were positive for Friedlander bacilli in both cases. Neither patient received chemotherapeutic or antibiotic agents, as these were not available at the time.

2. Friedlander bacillus meningitis should be treated promptly with sulfadiazine and streptomycin, since these drugs have been shown to have value, both in vitro and in vivo.

ADDENDUM

Since this report was submitted, S. S. Jacob and F. H. Top (Annals of Internal Medicine, 28:1003-1009, May, 1948) have reported seven cases of Friedlander bacillus meningitis with two recoveries. The patients who recovered had negative blood cultures and both seemed to be helped by sulfadiazine and penicillin.

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TABLE 2.—*The Effect of Treatment with Streptomycin on Experimental Infections in Mice with Micro-Organisms of the Friedlander Group.* (From Heilman[2])

Infecting Dose				Treatment with Streptomycin				
Experiment	Strain	No. of Times Lethal Dose	How Given	Units per Day	No. of Days	Inoculated Mice	Mice that Died	Mortality Per Cent
1	No. 575 Type Unk.	10,000	Intra-abdominally	185	3	8	0	0
				460	2	6	0	0
				None		14	14	100
2	No. 837 Type A	1,000	Intra-abdominally	500	3	20	1	5
				250	3	20	6	30
				None		20	20	100
3	No. 838 Type A	1,000	Intra-abdominally	500	3	15	0	0
				None		15	15	100
4	No. 837 Type A	100	Intranasally	500	3	10	8	80
				None		10	10	100
5	No. 837 Type A	100	Intranasally	500	7	15	2	13
				None		15	15	100

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CORRECTION:

In an article "Inflammation as Considered by the Radiologist," by Thomas M. Fullenlove, M.D., which was published in the August issue of CALIFORNIA MEDICINE, the word "leukotoxine" as it appeared several places in the text as well as in Chart 1 should have been "leukotaxine." The author of the article explains that leukotaxine, a word coined by Valy Menkin, is a combination of leukocyte and chemotaxis. Menkin's "Dynamics of Inflammation" is cited as authority.